# ACUTE AND CHRONIC MEDIASTINITIS

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# ACUTE AND CHRONIC MEDIASTINITIS

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Dedicated To
My Mother and Father
and To Beverly

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## INTRODUCTION

Mediastinitis may be defined as an inflammation of the areolar tissue and its blood supply lymphatics and nerve supply.[29] This inflammation may be acute or chronic, diffuse or localized, suppurative or nonsuppurative.

The purpose of this investigation is to review the clinical and pathological features of acute and chronic mediastinitis. All cases of mediastinitis identified at the Yale-New Haven Hospital during the period April, 1971 to June, 1977 were compared to the world's literature on mediastinitis to determine: (1) whether there has been any change in the incidence, etiologies, pathogenesis, pathology and signs and symptoms of mediastinitis and (2) what therapy is associated with the best prognosis.

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# I. NORMAL ANATOMY OF THE MEDIASTINUM

## NORMAL ANATOMY OF THE MEDIASTINUM

The mediastinum is the extrapleural subdivision of the thorax located between the pleural cavities. It extends from the sternum anteriorly to the vertebral column posteriorly. The upper limit is formed by the thoracic inlet and the lower border is represented by the diaphragm.[45, 16,31]

The mediastinum has been arbitrarily subdivided into several compartments. A plane which extends from the sternal angle to the fourth intervertebral disc subdivides the mediastinum into an upper and lower division. The upper division is the superior mediastinum. The lower division is further subdivided into three spaces---the anterior, middle and posterior mediastinum (See Figure 1).[31]

The superior mediastinum contains the thymus or its remnants, the innominate veins, the superior vena cava, the aortic arch and its major branches, the vagus nerves, the left recurrent laryngeal nerve, the phrenic nerve, the arch of the azygous vein and the upper segment of the hemiazygous system, the trachea, the esophagus and the thoracic duct. Lymph nodes in the superior mediastinum are found adjacent to the large vessels, the trachea and the spine.

The anterior mediastinum is a shallow space bounded anteriorly by the sternum and the transverse thoracic muscles and posteriorly by the pericardium. Anterior mediastinal contents include the lower portion of the thymus, sternopericardial ligaments, left internal mammary vessels and lymph nodes.

The middle mediastinum is bounded primarily by the pericardial sac and tracheal bifurcation. Contents of the middle mediastinum are all structures enclosed in the pericardium, the roots of the lungs, the pulmonary arteries and veins, the phrenic nerves and the tracheobronchial lymph nodes (See Figure 2).

The posterior mediastinum extends from the posterior aspect of the pericardium to the spine. Posterior mediastinal contents are the esophagus, descending aorta, the vagus nerves, the thoracic duct, the sympathetic chains, paravertebral lymph nodes and the azygous and hemiazygous venous systems.

The anatomical classifications presented so far are purely artificial ones. The superior mediastinum communicates widely with the other mediastinal compartments as well as with the visceral compartments of the neck.

Radiographically in the frontal view, the right border of the mediastinum is composed of the right innominate vein, the superior vena cava and the right heart border. The left mediastinal margin is formed by the left subclavian vessels, the aorta, pulmonary artery and left cardiac border.[31]

The very shallow anterior mediastinum is visualized radiographically only with great difficulty. This potential space is best demonstrated on chest radiographs taken in the lateral projection on deep inspiration and in recumbency.[31]

Roentgenographic findings indicate that the posterior mediastinum is located somewhat left of the mid-line and measures not more than two cm. wide.

The mediastinum is a potential extrapleural space in the thoracic cavity through which infection can spread from the neck and abdomen. The anatomy of the fascial planes, although not inviolate barriers to the spread of infections,

do influence the early spread of infections and are important in understanding the evolution of signs and symptoms.[16,29]

In the neck there are three important spaces which are potential pathways for the spread of infection (See Figure 3): (1) The retropharyngeal or prevertebral space is a potential space bounded by the prevertebral fascia posteriorly and the buccopharyngeal fascia anteriorly. Laterally these layers fuse as they join the carotid sheath.[16] The retropharyngeal space is continuous from the base of the skull to the level of the tracheal bifurcation. (2) The pretracheal space is bounded posteriorly by the pretracheal fasica and anteriorly by the anterior layer of the deep cervical fascia; it is limited by attachments to the sternum and the pericardium. (3) The visceral space is the area between the retropharyngeal and the pretracheal spaces and contains the trachea, esophagus, vagus and recurrent laryngeal nerves and the thyroid and parathyroid glands.

Access to the mediastinum from the retroperitoneum and lumbar regions is via unobstructed diaphragmatic hiatuses. Thus, the only tissue barrier from the pharynx to the peritoneum is the loose areolar connective tissue which is continuous throughout.[45]

# II. ACUTE MEDIASTINITIS

## INTRODUCTION

Kornblum and Osmond[29] and Neuhof[41] classified acute mediastinitis into two broad categories: (1) suppurative mediastinitis and (2) non-suppurative mediastinitis. Each of these categories was further divided into localized and diffuse involvement. The importance of acute mediastinitis is largely dependent upon whether or not suppuration occurs. Acute non-suppurative mediastinitis is a frequent complication of pleural and lung infections, but the mediastinitis is often not recognized clinically because it is over-shadowed by the primary disease.[29] Usually there are no specific symptoms and the mediastinitis subsides without serious sequelae provided the primary disease responds to treatment. Acute nonsuppurative mediastinitis is actually a reactive mediastinal cellulitis, that may be identified on chest radiographs.

Acute suppurative mediastinitis is a serious clinical entity and therefore will be the area of emphasis for acute mediastinitis.

# ETIOLOGY

## Esophageal Perforations

Perforation of the esophagus is a well documented complication of paraesophageal surgery. Local damage may occur secondary to direct surgical trauma or ischemic necrosis. Intercostal drainage tubes may produce esophageal perforation as a result of ischemic necrosis. Damage and perforation of the esophagus have been reported as complications of balloon tamponade for bleeding varices, of Negus bag dilitation for achalasia and dilitation of the thoracic esophagus for stricture of this organ.[26]

Involvement of the retrovisceral space with extension into the posterior mediastinum is the commonest pathway for spread of infection from perforation of the cervical esophagus since most perforations in this segment are through the posterior wall.[45]

Spontaneous rupture of the esophagus, a disease which usually follows a bout of vomiting, was reported as long ago as 1724.[37]

## Post-Surgical Anastamotic Leaks

When mediastinal infection occurs after esophageal and proximal gastric surgery, it is thought to result from imperfect healing and disruption of the suture line rather than due to immediate contamination.[16]

## Extension of Cervical Infections

Most cases of mediastinitis occurring secondary to extension from the neck are located in the superior and posterior mediastinum, not the anterior mediastinum, due to the structure of and the courses taken by the fascial planes.[12,14,31,53]

However, in fulminating infections of the mediastinum, tissue planes are no longer inviolate barriers to spread to nearby compartments.[45]

## Tracheal Perforation

Infections involve the pretracheal space from perforations of the tracheal wall as well as from the lateral pharyngeal spaces (the pyriform sinus).[53] Such infections extend into the anterior mediastinum. Fluctuations in negative intrathoracic pressures during normal respiration tend to draw the contents of fascial spaces into the mediastinum.[10,16,53]

## Surgical Wound Infections

Cervical and mediastinal incisions occasionally become infected with extension to the anterior mediastinum, especially median sternotomy and occasionally tracheostomy incisions.[14,16] Predisposing factors in median sternotomy infections reported by many authors include pneumonitis before or after surgery with extension to the mediastinum, tracheostomy, prolonged cardiac bypass time, postoperative corticosteroid therapy, closed chest massage and poor postoperative cardiac output.[9,34,57] Protein denaturation and destruction of defense mechanisms by bypass machines may play a role in the increased incidence of infections.[9,24]

On rare occasions, operations on the thyroid, laryngectomy and mediastinoscopy have been followed by acute mediastinitis.[16]

## Extension of Abdominal Infections

Pancreatitis and subphrenic and retroperitoneal infections can extend into the mediastinum via the diaphragmatic hiatuses for the great vessels

and the esophagus. However, because of the severity of their clinical presentations, these extrathoracic infections are rarely permitted to progress to the stages of mediastinal involvement.[14,45]

Miscellaneous

Diabetes mellitus has not been established as a predisposing factor in these infections.[34]

## SYMPTOMS AND SIGNS

In 1936, Neuhof[41] described the classic presentation of acute mediastinitis as follows: recurrent chills, high fever, profound prostration, severe toxemia, and typhoid state. He pointed out that such a presentation is rare. In six of his 66 reported cases, the mediastinal infection was associated with no clinical symptoms and was a postmortem disclosure.

Other clinical features include chest or epigastric pain, respiratory distress, cough, tachycardia, dysphagia, swollen neck and neck vein distention.[1,16,45] Pain on swallowing and inspiration has been mentioned as an important early sign of mediastinitis.[13] Dysphagia, dyspnea, venous distention and edema of the head, thoracic wall and upper extremities reflect encroachment upon mediastinal structures and are late signs of acute mediastinitis.[13] Respiratory distress is usually a sign of pleural involvement.[45]

Soft tissue emphysema usually indicates perforation of the trachea or esophagus.[26,31,45] Mediastinal emphysema is an early and valuable sign of esophageal perforation. Subcutaneous emphysema extending into the neck may be seen later.[14]

Perforation of the cervical esophagus with leakage of contained materials characteristically produces neck pain; mid-esophageal perforation produces substernal pain; perforation of the lower esophagus produces epigastric pain which may result in the erroneous diagnosis of perforated peptic ulcer or acute pancreatitis.[1,37,45] Frequently the earliest symptom in esophageal rupture is pain on swallowing.[1] Localization of pain with swallowing often coincides with the site of esophageal perforation.[45]

Chest radiographic findings vary from normal in mild disease to the presence of diffuse mediastinal widening or a mediastinal mass due to abscess formation.[14,16,26,31] The chest film is often negative in phlegmonous mediastinitis and in mediastinal abscess located near the diaphragm although localized widening of the mediastinum is a frequent finding in abscesses as well as in phlegmons of the mediastinum.[41]

Lateral neck films demonstrate a widened retrotracheal space with or without fluid level in infections descending from the neck into the mediastinum.[31,41]

Unilateral or bilateral hilar and basilar pulmonary infiltrates develop as mediastinal infection extends. Also, pleural effusion may be detected.[16]

Any patient suspected of having suffered esophageal perforation should be promptly examined radiographically by a limited upper gastrointestinal series with a water soluble contrast agent.[23,26] If this study is negative, it should be repeated using barium. Radiographic signs include extravasation of contrast from the esophagus, air in the tissue planes of the neck and mediastinum, anterior displacement of the esophagus, widening of the mediastinal shadow, elevation of the diaphragm and obliteration of the costophrenic or the cardiophrenic angle.[1]

In a paper on spontaneous rupture of the esophagus, Movas[37] reported spontaneous emphysema at the root of the neck in 65% of cases. Also, chest radiographs were positive for pleural effusion or hydropneumothorax in 91% and mediastinal emphysema in 66%.

In patients with mediastinitis secondary to median sternotomy wound infections, the infection was heralded by purulent wound drainage and/or

sternal instability.[4,9,10,19,24,57] Other features include leukocytosis (greater than 10,000 white blood cells per $mm^3$) and fever (greater than $38.3^{o}$ C). Several authors report the onset of signs of infection at about two weeks postoperatively.[19,34] Chest radiographs in these patients may be normal or show mediastinal widening indistinguishable from routine post-operative changes.[9,10] Ultimately the diagnosis of mediastinitis is based upon drainage of purulent material from the mid-sternotomy wound, regardless of fever, leukocytosis and positive cultures which are not always present.[24]

## INCIDENCE

Acute suppurative mediastinitis has been considered an uncommon disease. However, this may be incorrect since the diagnosis is not made readily.[16] The advent of antibiotics has been reported to have decreased the incidence further.[13]

In 1936, Neuhof described 66 cases of acute mediastinitis, approximately 50% of which were due to instrument or foreign body perforation of the esophagus.[16,41] Another 25% of cases were due to nontraumatic infections in the cervical region.[10,41] Recently, Enquist et al.[10] stated that suppurative mediastinitis usually occurs secondary to esophageal perforation or median sternotomy incision infection.

Payne and Larson,[44] reported a 0.4% risk of esophageal perforation by instrumentation while Triggiani and Belsey[57] reported an incidence of 0.2% based on their large retrospective study of esophageal trauma. The global incidence of instrument perforation is impossible to ascertain since few of these catastrophies are ever reported.[58]

The incidence of mediastinitis secondary to median sternotomy wound infection is variously reported from 0.5 to 5%[9,19,57] In 1970 Jimenez-Martinez et al.[24] reported a 5% incidence of major mid-sternotomy infections in patients subjected to cardiopulmonary bypass and an incidence of 3.6% in those which bypass was not employed.

In a series of 1,042 median sternotomy incisions reported by Engelman et al.,[9] 1.6% of the patients developed mediastinitis. Among those undergoing coronary revascularization, the incidence was 2.7%; among those undergoing valve replacement, the incidence was only 1.4%.

The incidences of mediastinitis secondary to metastasis from distant infectious foci and major upper and lower respiratory tract infections have always been regarded as relatively rare and have become rarer since the

advent of antibiotics.[13,29,45,53]

BACTERIOLOGY

Remarkably few of the articles in the literature on acute mediastinitis of nonsurgical origin mention anything about the infecting organisms.

In 1934, Kornblum and Osmond[29] identified the streptococcus and staphylcoccus as being most common with occasional infections due to pneumococcus or diptheroid bacillus. The disease may also be the result of infections by tubercle bacillus, treponema pallidum and actinomycosis.

In 1936, Neuhof[41] identified the hemolyte streptococci as the organisms most commonly isolated from nonperforative suppurations of the mediastinum.

Enquist et al.[10] in 1976 in a paper on nontraumatic mediastinitis reported B-hemolytic streptococci as the most commonly isolated organism from mediastinitis secondary to infection of the throat and neck regions. Also, odontogenic infections may result in a descending necrotizing mediastinitis caused by one or more species of gram-negative aerobic and anaerobic bacteria, usually anaerobic streptococcus and bacteroides.

Abundant information exists on the bacteriology of mediastinitis secondary to median sternotomy infection. In Jimenez-Martinez's[24] report on 19 patients with infected mid-sternotomy incisions, cultures were positive in only five: staphylococcus aureus in three; klebsiella in one; pseudomonas aeruginosa in one.

Engelman et al.[9] in a paper on 17 patients with mid-sternotomy wound infections found multiple organisms in seven patients. Organisms isolated were as follows:

| ORGANISMS | NUMBER OF PATIENTS |
|---|---|
| Staphylococcus Albus | 6 |
| Klebsiella | 6 |
| Candida | 4 |
| E. Coli | 3 |
| Pseudomonas | 2 |
| Streptococcus Fecalis | 2 |
| Proteus Mirabilis | 2 |
| Citrobacter | 1 |
| Serratia | 1 |

The absence of staphylococcus aureus in this series is attributed to the use of prophylactic antistaphylococcal antibiotics.

Thurer et al.,[56] in a series of 14 cases, obtained positive cultures from all patients. About one-third of these had more than one organism recovered on culture which included staphylococcus aureus (both coagulase positive and coagulase negative), E. Coli, Klebsiella, Enterobacter, Pseudomonas, Serratis, Proteus and alpha and beta-streptococcus. All patients in this series received prophylactic antibiotics preoperatively, usually a cephalosporin.

Grmoljez et al.[19] reported similar bacteriologic results. Staphylococcus aureus was the most common isolate. All patients received preoperative prophylactic antibiotics.

In 1976, Cerat et al.[5] reported a unique bacteriologic finding--a case of bacteroides fragilis infecting a mid-sternotomy wound. They also reported 95 patients with positive cultures out of 131 total sternotomy infections. In those positive cases, enteric gram-negative bacilli were most common.

## TREATMENT

In general, treatment of mediastinitis consists of surgical drainage of the mediastinum and appropriate antibiotic therapy.[16,45]

### Esophageal Perforation

In 1936, Neuhof[41] concluded that in cases of esophageal perforation, external operation consisting of exposure of the tear and drainage of the area is indicated urgently, regardless of the site of the lesion. However,

it was not until 1947 that Barrett performed the first successful thoracotomy and repair of an esophageal tear.[37]

Since Neuhof's conclusions on treatment, it has become evident that treatment can be safely tailored to the particular site of the esophageal lesion. Many perforations of the cervical esophagus (due to instrumentation or foreign body) may respond to cessation of swallowing, rest and antibiotics without developing mediastinitis. However, a more reliable form of management is prophylactic cervical mediastinal drainage. No attempt need be made to suture closed the rent.[1,26,45]

Esophageal perforations proximal to unrelieved esophageal obstruction and postemetic esophageal rupture (Boerhaave's Syndrome) are serious problems and require early surgical intervention with debridement, closure by suturing and appropriate drainage of the pleural and peritoneal spaces. Gastrostomy for fluid and food administration is essential until the esophagus has healed.[1,26,37,45] Antibiotic therapy is continued until all signs of systemic reaction to infection have subsided.[1]

Movas (1966)[37] proposed that in cases of spontaneous esophageal rupture which are diagnosed late (2-3 days after rupture) no operation is necessary if the patient is in good general condition and is without evidence of increasingly enlarging mediastinal abscess or empyema.

## Retropharyngeal Abscess

Retropharyngeal abscess may be treated by drainage alone.[26] If there is extension of suppuration into the mediastinum, a tract can be laid open adequately from the neck to as far as the body of the third or fourth thoracic vertebra. Drainage through the neck will not be adequate below this level and posterior mediastinotomy should be performed.[41,45]

Mid-Sternotomy Incision Infection

The treatment of mediastinitis secondary to infected mid-sternotomy incisions is quite different from the treatment discussed above. The original treatment of this problem consisted of debridement of the infected areas and open drainage of the mediastinum. Problems with this treatment are that exposed pleural cavities prevent adequate cough reflex, impair ventilation and prolong recuperation.[9,24]

In 1960, Spencer developed the concept of debridement followed by closed mediastinal irrigation with antibiotic solution,[9] a concept which is still accepted.[24]

As soon as infection is recognized, the patient should be taken to the operating room and the entire sternotomy wound reopened. The sternotomy should be debrided down to healthy bone and necrotic debris within the mediastinum should be removed.[4,24,57] Gross pus should be sought and drained. The entire wound is then thoroughly irrigated with either a dilute betadine solution or an antibiotic solution consisting of neomycin or a combination of neomycin and bacitracin. Kanamycin has also been used as an irrigant. Next, drainage and irrigation tubes are placed in appropriate positions within the mediastinum and the pleural spaces if their integrity was violated.[4,19,24,57] The sternum is then closed primarily with wire sutures followed usually by primary closure of the overlying skin.[4,57] Drainage tubes are connected to closed suction devices and irrigation is begun immediately post-operatively.

Bryant et al.[4] described the use of a neomycin-polymyxin-bacitracin solution which was infused into the mediastinum at a rate of 50-100 ml per hour. It usually consisted of two grams of neomycin and 100,000 units of bacitracin per liter of saline. The solution was infused for 10-14 days

followed by gradual removal of the mediastinal tubes. Appropriate systemic antibiotics also were given intravenously over a similar period of time. Although there is no control information available, healing of the sternum and the absence of fever are the best indicators that treatment is successful.

Engelman et al.[9] described the use of 2 grams of neomycin and 50,000 units of bacitracin per liter of physiologic saline which was infused continuously for 5 to 21 days. The chest tubes were removed up to six days after cessation of irrigation.

Thurer et al.[57] described the use of continuous irrigation of the mediastinum with a 0.5% solution of betadine in saline which was infused continuously at 50-100 cc per hour for 6-7 days. Broad spectrum antibiotics were given intravenously beginning at the time of debridement and were continued until all drainage tubes were removed. Thurer also pointed out that while mediastinal lavage with antibiotics is highly efficacious, the patient is predisposed to antibiotic toxicity and antibiotics may facilitate development of chronic infections of the sternum and costal cartilages by candida albicans. Persistent osteomyelitis and osteochondritis due to pseudomonas has also been reported. Betadine, on the other hand, has broad bactericidal and fungicidal properties and lacks observed toxicity in the concentrations used.

In summary, a variety of therapeutic methods have proved effective.

## MORBIDITY AND MORTALITY

### Esophageal Perforation

Many authors report a good prognosis for recovery in cases of esophageal rupture which are treated promptly by surgical techniques.[1,23,37,58] Without surgical drainage this disease is usually fatal within hours. The mortality rate increases in proportion to the time interval between perforation and surgical intervention.[23,42] Spontaneous cure following rupture of a mediastinal abscess has, however, been reported on rare occasions.[13,41]

In a report on the diagnosis of esophageal tears, James et al.[23] mention that more than 70% of untreated patients succumb to septic gangrenous mediastinitis, pleuritis, pyopneumothorax and empyema within several days after the insult.

In a report on morbidity and mortality of esophageal perforation, Keighley et al.[26] reported 33 patients seen over a 20 year period. In this series, endoscopic perforation in patients with neoplastic disease of the esophagus had a 100% mortality despite early diagnosis and immediate resection of the growth. In contrast, mortality was only 20% for perforation with underlying benign disease of the esophagus. Mortality was 8% after spontaneous perforation. Although mortality after spontaneous perforation was low, the morbidity (empyema, esophageal fistula, local secondary abscess) of survivors was considerably higher than in those with instrument perforation. It was assumed that the high incidence of complications after spontaneous rupture occurred secondary to leakage from the repair site as identified by contrast radiography.

INCIDENCE AND MORTALITY
OF
ESOPHAGEAL PERFORATIONS 1951-71 (33 CASES) [26]

| INCIDENCE | | | MORTALITY | |
|---|---|---|---|---|
| A. Endoscopic perforations | | 16 | 8 | (50%) |
| for carcinoma | 6 | | 6 | (100%) |
| for benign pathology | 10 | | 2 | (20%) |
| B. Foreign body trauma | | 1 | 0 | |
| C. Following thoracotomy | | 3 | 0 | |
| D. Spontaneous rupture | | 12 | 1 | (8%) |
| TOTALS | | 32 | 9 | (28%) |

MORBIDITY OF ESOPHAGEAL PERFORATION IN SURVIVORS [26]

| | | INCIDENCE OF POST-OPERATIVE COMPLICATIONS |
|---|---|---|
| Endoscopy for Benign Pathology | 8 | 3 |
| Foreign Body | 1 | 1 |
| Following Thoracotomy | 3 | 3 |
| Spontaneous Rupture | 11 | 9 |
| TOTAL | 23 | 16 |

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INSTRUMENTAL PERFORATION

| | | MALIGNANT (N = 6) | BENIGN (N = 10) |
|---|---|---|---|
| Avg. Age | | 72 years | 50 years |
| Sex Group | | 83% Male | 90% Female |
| Sites: | Cervical | ------- | 4 40% |
| | Thoracic | 6 100% | 6 60% |
| Diagnosis: | Less than 24 hrs. | 4 66% | 6 60% |
| Treatment: | Resection | 2 | 1 |
| | Repair | - | 4 |
| | Conservative | 4 | 6 |
| MORTALITY | | 6 100% | 2 20% |

Cervical Suppurations

In 1938, Pearse reviewed 110 cases from the literature on mediastinitis following cervical suppuration.[42] With operative intervention he reported a 35% mortality as compared to an 85% mortality when operation was not performed.

Mid-Sternotomy Incision Infections

Engelman et al.[9] reported 1,042 median sternotomies done over a two year period in which 17 patients developed mediastinitis. Two of the 17 died of candida albicans mediastinitis and were symptom free and thus were not treated. Five of the 15 treated patients died.

When mediastinitis follows open heart surgery for coronary revascularization, involvement of the grafts by the infectious process may result in graft dehiscence or graft closure.

Macmanus and Okies[34] stated that an increased incidence of prosthetic endocarditis has been seen in mediastinal infection.

# III. CHRONIC MEDIASTINITIS

## INTRODUCTION

Some attribute the first description of mediastinal fibrosis to John Hunter in 1757; others prefer the description by Hallet[57] in 1848.

Chronic affections of the mediastinum can be divided into two broad categories: (1) mediastinal granulomata, a term applied to caseous or fibrocaseous lymph nodes or masses found within the mediastinum and (2) fibrosing mediastinitis which is now believed to be a late stage of mediastinal granuloma.[17,20,22,52,60] These two groups are not separated in all articles in the literature discussing chronic mediastinitis.

The first group, granulomatous mediastinitis is massive coalescent adenitis with surrounding fibrosis limited to that of ordinary encapsulation. In contrast, fibrosing mediastinitis refers to prominent fibrosis with a less conspicuous or absent granulomatous component.[17]

PATHOLOGY

Mediastinal fibrosis is a rare condition characterized by formation of dense fibrous tissue which engulfs and destroys surrounding structures. The condition usually affects the superior mediastinum, especially in the region of the superior vena cava (SVC) at or near the tracheal bifurcation of the pulmonary hilum. Less commonly, the fibrosis affects other mediastinal structures.[52,59]

The fibrosis consists of a woody, white fibrous tissue mass usually located on the right side of the mediastinum. It may take the form of a flat, plaque-like tissue or nodular, tumor-like masses. Sometimes the tissue is clearly circumscribed, but in the majority of cases the limits are poorly defined. The mediastinal pleura in the vacinity is thickened but the lung is rarely invaded. The aorta, trachea, esophagus, nerves and thymus are commonly found adherent to the fiberoptic masses and to each other. Much less commonly are these organs functionally impaired and often no referable clinical signs of their involvement develop.[2,27]

Among the 103 cases of granulomatous mediastinitis reported in the literature between 1942 and 1969, intrusion upon adjacent structures occurred in 27 (26%).[52] The structures involved were are follows: SVC (14), esophagus (9), tracheobronchial tree (2), pulmonary vein (1), tracheoesophageal fistula (1). Occlusion of the inferior vena cava by mediastinal fibrosis has been reported but is extremely rare.[52]

Histologically the abnormal tissue consists of dense collagenous fibrous tissue that is almost acellular without evidence of caseation,liquefaction or granuloma formation.[2,17] In the early or granulomatous stage of chronic mediastinitis, the basic pathologic finding is caseous granulomatous adenitis surrounded by a fibrous capsule of varying thickness.[17,56]

The histologic appearance of mediastinal fibrosis has suggested to several investigators that mediastinal fibrosis is related to other fibrosing diseases such as retroperitoneal fibrosis, fibrous thyroiditis, pseudotumor of the orbit, Dupuytren's contracture of palmar fascia, Peronie's disease of the penis and sclerosing cholangitis. At least six cases have been reported in which retroperitoneal fibrosis and mediastinal fibrosis have coexisted.[40,52]

The critical determinants of the disease process of fibrous mediastinitis are the degree of fibrosis and the location of the lymph node or nodes acting as a point of origin of the fibrosis.[17] The final distribution of fibrosis within the mediastinum appears to be along the lymphatic pathways draining the lungs suggesting that the pulmonary parenchyma is the most likely site of the initial infection.[22] Lymph channels from both lungs flow to the hilar and subcarinal lymph nodes and then toward the right paratracheal chain. These are the three main areas involved. (See Figures 4 and 5). Extensive fibrosis within the right paratracheal area always involves the SVC and azygous vein. Subcarinal fibrosis results in three different patterns of major organ involvement depending on the direction in which fibrosis extends. Anterior extension involves the pulmonary veins; posterior extension involves the esophagus; lateral extension involves the main bronchi and pulmonary arteries. These findings may be observed singly or in combination.[17]

If the site of venous obstruction is above the SVC, one innominate vein (usually the left) is spared. In this instance clinical signs may not be diagnostic because the obstructed blood may be shunted through the cross-circulation in the neck.[2] (See Figures 6-8)

When the obstruction includes the SVC, but is above the azygous vein, the collateral veins on the front of the chest do not become prominent because the superior intercostal veins carry the blood into the azygous and back to the heart.[2]

If the azygous vein itself is blocked as well as the SVC, the collateral veins both inside and outside the chest become dilated and they carry all the blood down to the inferior vena cava.

The fact that the azygous and the hemiazygous venous systems contain no valves is important because the direction of the flow can be reversed where necessary. In comparison, the superficial and the remaining deep venous collaterals must distend until their valves become incompetent before they can carry a reversed bloodflow.[2]

In those rare cases of pulmonary artery obstruction by mediastinal fibrosis, an exuberant collateral bronchial circulation not only provides nutrition to the lung, but may also permit perfusion and partially restore respiratory function.[7]

# ETIOLOGY AND PATHOGENESIS

## ETIOLOGIES

During the latter part of the 19th and the early 20th centuries, it was customary to ascribe chronic mediastinitis to tuberculosis or syphilis.[17,40] However, the diagnosis of tuberculosis was based solely on the presence of either caseation or calcification within the lesion, both of which are nonspecific findings. The diagnosis of syphilis was based upon the presence of a positive serology or evidence of syphilis elsewhere in the body.

In an extension review of superior vena cava obstruction in 1936, Ochsner and Dixon[43] attributed 28 of their 120 cases (23%) to mediastinal fibrosis. Etiologies included tuberculosis (10), syphilis (11), trauma (1), bacterial infection (1) and idiopathic (5).

In 1937, Keefer[25] listed the following causes of chronic fibrous mediastinitis: tuberculosis, syphilis, mycotic infection, rheumatic fever, pyogenic infection, pneumoconioses and chronic paraganomiasis.

In 1944, Blades and Dugan[3] reported the microscopic demonstration of acid-fast organisms within a resected mediastinal granuloma, providing the first recorded case in which a specific agent was demonstrated.

Histoplasmosis was first considered a causative factor in mediastinal granuloma by Samson in 1950.[47] Three years later, Puckett[46] reported histologic demonstration of the yeast cells of Histoplasma capsulatum in resected pulmonary granulomatous nodules and hilar lymph nodes. His discovery followed the development of the periodic acid-Schiff stain and later a methenamine silver stain, both of which were effective in demonstrating this organism. Straub and Schwartz[54] described the primary lesion of histoplasmosis and demonstrated the persistence of stainable organisms in healed and even calcified remnants in pulmonary parenchyma and hilar and mediastinal lymph nodes.

In 1958, Barrett[2] pointed out that cultures from fibrotic mediastinal tissues removed at surgery are always sterile. However, in that same year, Peabody et al.[45] reported five cases of typical encapsulated granuloma in which H. capsulatum was demonstrated histologically by careful restaining previously resected, "nonspecific" granulomas.

The cause of most cases of mediastinal fibrosis is unknown despite extensive bacteriologic, serologic and pathologic study.[40] As it became evident that a positive identification was not so easily made, the number of cases reported as idiopathic increased from 23% in 1934 to 65% in 1946 to 96.1% in 1969.[52] Of 77 cases of fibrous mediastinitis reported by Schowengerdt et al in 1969,[51] the presence of organisms was demonstrated in only three cases (3.9%). Histoplasmosis was the cause in two cases and acid-fast organisms in the third. However, these authors made a definitive etiologic diagnosis in 33 of 103 cases of granulomatous mediastinitis (32%). Etiologies of granulomatous lesions included histoplasmosis (14), tuberculosis (13), sarcoidosis (4), silicosis (1), and nocardia (1). These investigators suggested that histoplasmosis probably constituted the etiologic agent in a much higher percentage of mediastinal granulomas than their figures indicated since many previously unproved cases proved later to be secondary to H. capsulatum histologically.

In 1972, Goodwin et al.[17] reported 38 cases of mediastinal granuloma of known etiology; 26 were related to healed histoplasmosis and 12 were due to mycobacteria in fection. H. capsulatum was identified by demonstration of morphologically typical yeast cells. In no instance was the organism recovered by culture. Mycobacteria were identified by acid-fast stains and in four of the 12 cases, mycobacteria were recovered by culture. Thus in the United States, tuberculosis and histoplasmosis are the two most frequently suspected diseases in chronic mediastinitis.[16,56]

Pathogenesis

Organisms enter the mediastinum via lymphatics, necrotic lymph nodes, empyema or from osteomyelitis of vertebrae, ribs or sternum.[16] Chronic mediastinitis has complicated pulmonary resections for tuberculosis and various mycoses.[32] Acute suppurative mediastinitis rarely produces chronic fibrous mediastinitis.[16]

Although the pathogenesis of fibrosing mediastinitis is unknown, a hypothesis is that it represents some type of immunologic disturbance in response to infection.[16]

In 1974, Kittredge and Nash[27] suggested sclerosing mediastinitis, retroperitoneal fibrosis, sclerosing cholangitis, Reidel's thyroiditis and pseudotumor of the orbit all represent manifestations of a common disease process having perhaps a systemic basis since these entities have been known to occur together in the same patient. These authors reported five cases of chronic fibrous mediastinitis in which no etiologic agent could be identified. They suggested the following possible etiologies: (1) drug sensitivity and (2) an underlying auto-immune disease or collagen disorders such as systemic lupus erythematosus, poly-arteritis nodosa, rheumatoid arthritis and Raynaud's phenomenon. However, there are no reports of any case of chronic mediastinitis associated with any of the auto-immune collagen disorders list above.

## SYMPTOMS AND SIGNS

The most common clinical symptoms of mediastinal granulomas are cough, chest pain, dysphagia and hemoptysis. Many patients are asymptomatic.[56] A right paratracheal or hilar mass on chest radiograph is common. The symptoms of granulomatous mediastinitis are directly related to the severity with which the adjacent structures are involved.[52]

The most common clinical feature of fibrosing mediastinitis is superior vena cava (SVC) obstruction of insidious onset and associated with few constitutional symptoms.[25,40] Signs and symptoms of SVC obstruction include: (1) headache, (2) vertigo, (3) tinuitus, (4) dyspnea, (5) chest pain, (6) hoarseness, (7) hemoptysis and (8) pitting edema and cyanosis of the face, neck, arms and upper chest.[2] The first symptoms are often intermittent, but there are records showing that severe and even fatal SVC obstruction can overwhelm a patient in a few hours. More commonly, the illness is slowly progressive over several years.[2,35,40]

Radiographically, the mediastinum may be broadened by distended parts of the azygous and the hemiazygous veins.[2] Also, increased pulmonary vascular markings may be present, especially in the upper lobes.[27] Pleural effusions can occur and edema of the glottis has been described.

In addition to SVC obstruction, clinical manifestations of major airway obstruction and esophageal obstruction have been reported.[22,60] Goodwin et al.[17] reported bronchial obstruction as the most common symptomatic manifestation of fibrous mediastinitis among the 38 cases reported. 29% of the cases demonstrated evidence of bronchial involvement by mediastinal fibrosis manifested by severe cough, hemoptysis, dyspnea, wheezing and episodes of obstructive pneumonitis. 50% of their cases were asymptomatic and the remainder were symptomatic of SVC obstruction.

Although SVC and bronchial obstruction are the most common complications of fibrosing mediastinitis, in isolated cases, involvement of the pulmonary veins, inferior vena cava, thoracic duct and pulmonary arteries have been reported.[7] In 1971, Yacoub and Thompson[58] described idiopathic pulmonary hilar fibrosis, a form of fibrosing mediastinitis which limits itself to the pulmonary hilum and its associated structures. This syndrome is characterized by repeated hemoptysis which may be associated with shortness of breath, pulmonary hypertension, pleural thickening and decreased size of one lung, segmental narrowing of the pulmonary arteries, veins or both. They classified this syndrome into two major types: In type I, the pulmonary arteries are affected; in type II, the pulmonary veins are affected. In type I, hemoptysis is probably secondary to exuberant bronchopulmonary anastomoses. The chest radiograph shows a shift of the mediastinum to the affected side, decreased size of the pulmonary artery shadow and pleural thickening. Pulmonary hypertension is present when both main branches of the pulmonary arteries are affected or when the pulmonary veins are involved. Pleural thickening is related to repeated chest infections and development of collaterals between the lungs and the chest wall. In type II, severe hemoptysis may occur secondary to pulmonary capillary hypertension. Cardiac catheterization shows increased pulmonary artery pressure which may equal systemic arterial pressure. Differential lung function tests show evidence of obstructive and restrictive disease of the affected lung. Also, diffusing capacity of that lung is decreased.

## DIAGNOSIS

When signs and symptoms of SVC obstruction are noted, the diagnosis is confirmed by venograms of the mediastinal veins. Such studies will show a plethora of dilated veins, the majority of which are never seen by venogram except when the SVC is obstructed.[2]

Since venography is not without mobidity and mortality (excessive bleeding from puncture sites in the presence of elevated venous pressures and local thrombosis or phlebitis secondary to slowed flow rates of venous blood), Lokich and Goodman[32] believe that in the presence of symptoms of SVC obstruction, venography is not warranted and that the diagnosis should be a clinical one.

Plain chest radiographic findings are nonspecific. They may be normal or may demonstrate enlarged lymph nodes, widening of the mediastinum, pleural thickening and calcification in the lungs, hilar lymph nodes or pericardium.[16,17]

A positive histoplasmin skin test and caseating granulomata on histologic examination may aid in distinguishing mediastinal granuloma and fibrosis from primary and secondary carcinoma, Hodgkin's disease and fibrosarcoma of the mediastinum.[27,57] Although typical histoplasma organisms can often be demonstrated on stained histologic sections, attempts to culture the organisms invariably have failed. It has been suggested that this organism is in an inactive stage.[56]

Routine blood tests, sputum cultures, complement-fixation and immuno-diffusion tests for histoplasmosis are generally negative in their results and so have not been helpful in establishing a diagnosis.[56] Bronchoscopy, esophagoscopy, bronchograms, bronchial biopsy and supraclavicular lymph node biopsy have been of no value in the diagnosis of granuloma or fibrosis since their results are consistently negative.[56]

INCIDENCE

Chronic mediastinal fibrosis most commonly affects the 20-45 year-old age group and women and men are about equally affected.[22,56,59] Descriptions of this disease date as far back as 1855. The cases were all associated with SVC syndrome and the etiology was assumed rather than proven.[36,52] Remarkably, there are no reports in the available literature of the incidence of mediastinal granuloma and fibrosis in the general population. However, in several large series of SVC obstruction, mediastinal fibrosis has been incriminated as the cause in from 10-23% of cases.[36,44,50,59] An unpublished review of a 10 year experience with histoplasmosis reveals that approximately eight percent of patients will show some evidence of fibrosing mediastinitis.

Between 1942 and 1969, 103 granulomas of the mediastinum and 77 cases of fibrosing mediastinitis were reported in the literature.[52] Schowengerdt et al.[51] stated that perhaps a reliable estimate is that granulomatous and fibrous mediastinitis account for approximately 10% of all primary mediastinal masses. However, their estimate was not based upon any particular series of cases and thus is of dubious validity. Approximately six percent of all mediastinal tumors treated surgically are mediastinal granulomas.[56]

TREATMENT

In 1942, Grace[18] reported the first surgical resection of an encapsulated mediastinal granuloma. Although surgical management of vascular obstruction due to chronic mediastinitis may be associated with an improved prognosis of relief of obstruction is feasible, it has become wident that the cornerstone of treatment in this disease is basically symptomatic and supportive.[7,16]

Effler and Groves[8] advocated that surgery not be done when the clinical picture is suggestive of benign SVC obstruction secondary to chronic granulomatous inflammation. These investigators noted that surgery has not improved long-term results since these patients had a remarkable good long-term prognosis without surgery. They suggested that patients with mediastinal disease still in the granulomatous stage without evidence of SVC obstruction should undergo surgical extirpation of the granuloma to prevent later development of SVC obstruction. In their experience with patients after prophylactic surgery, none developed SVC obstruction; neither was there dissmination of infection because of surgery.

Hewlett et al.[22] pointed out that due to many technical difficulties with surgery in the late stages if chronic fibrous mediastinitis, surgery should be limited to decortication of vital structures such as the major airways and large mediastinal vasculature.

Medical treatment is of no avail in chronic granulomatous or fibrosing mediastinitis. Anti-tuberculosis and anti-syphilis treatment has been ineffective. Cortisone and heparin are disappointing and radiotherapy is harmful.[2] Although corticosteroid therapy has been reported to be highly effective in isolated case reports, prospective, controlled studies are lacking.[7] Strimlan et al.[45] believe corticosteroids may be of value in preventing scarring and vena cava or bronchial obstruction in surgically proven, culture negative cases with extensive fibrosis.

Experience with pulmonary histoplasmosis has indicated little or no benefit from treatment with amphotericin B unless there is clearly active progressive infection.[7]

During the acute phase of SVC obstruction due to mediastinal fibrosis, symptomatic treatment of vascular congestion with a low sodium diet, diuretics and steroids has been advocated.[35]

## MORBIDITY AND MORTALITY

The prognosis of chronic fibrous mediastinitis and its associated complications (SVC, esophageal and major airway compression) is usually good.[2,35,56] Affected patients have been reported to survive 20-50 years after the onset of symptoms. Although mediastinal fibrosis is a self-limiting process which tends to remain stationary after the first few months in the disease course, permanent interstitial pulmonary fibrosis and progressive secondary changes in pulmonary arterioles may complicate pulmonary venous obstruction.[17,59] This was first described by Andrews in 1957.

# IV. ACUTE MEDIASTINITIS

## Materials and Methods

Retrospectively, all identifiable cases of acute mediastinitis occurring at the Yale-New Haven Hospital between April, 1971 and June, 1977 were sought via an intensive search of the medical records library. Two classifications were specifically relied upon: (1) mediastinitis (519.9) and (2) esophageal rupture (530.4). Additionally, members of the following departments at the Yale-New Haven Hospital were interviewed to determine if additional cases of acute mediastinitis could be found: (1) Radiology, (2) Infectious Diseases, (3) Gastroenterology, (4) Cardiothoracic surgery, and (5) Otolaryngology. All records received were reviewed for a diagnosis of acute mediastinitis and its associated signs and symptoms, radiographic findings, bacteriology, pathology, treatment and morbidity and mortality.

## Median Sternotomy Wound Infection

Ten cases of acute mediastinitis secondary to median sternotomy wound infection were found at the Yale-New Haven Hospital between the period April, 1971 through June, 1977. These cases represent 50% of all cases of acute mediastinitis at this hospital during the stated period.

### DEMOGRAPHIC DATA

| No. | PATIENT | SEX | AGE (YRS.) |
|---|---|---|---|
| 1 | W.B. | M | 58 |
| 2 | W.C. | M | 64 |
| 3 | G.F. | F | 47 |
| 4 | M.G. | M | 73 |
| 5 | W.V. | M | 71 |
| 6 | G.C. | M | 64 |
| 7 | M.L. | F | 62 |
| 8 | A.S. | F | 60 |
| 9 | A.B. | M | 60 |
| 10 | R.G. | M | 52 |

CLINICAL SYMPTOMS

The only evidence of infection in patient no. 1 was sternal dehiscence, occurring nine days after coronary artery bypass surgery.

Two days after coronary artery surgery, patient no. 2 experienced tachypnea, diaphoresis, cyanosis and fever to $104^{o}$ F. All but the latter resolved themselves. Finally, on the 16th postoperative day, he drained frank pus from the upper corner of his sternotomy incision

Patient no. 3 underwent open-heart surgery for repair of a congenital atrial septal defect and pulmonary stenosis. On the 23rd postoperative day, drainage from the sternotomy incision was noted. This was preceeded by a history of fever to $104^{o}$ F two days prior to drainage of the wound.

Fifteen days after coronary artery bypass surgery, patient no. 4 experienced drainage from the sternotomy wound and frank dehiscence from the sternum. There was also associated fever to $103^{o}$ F.

Patient no. 5 underwent resection of an ascending thoracic aortic aneurysm and aortic valve replacement. On the eighth postoperative day, the patient experienced respiratory distress of uncertain etiology. On the 28th postoperative day, he experienced dehiscence of the sternum near the sternal notch.

Patient no. 6 underwent coronary artery bypass surgery which was followed by purulent discharge from the sternal wound 16 days postoperatively and fever to $102^{o}$ F.

Patient no. 7 underwent coronary artery bypass surgery and was discharged three weeks later after an uneventful hospital course. However, eight days after discharge, the patient noted an erythematous skin lesion at the upper pole of the sternotomy incision. Two days later, purulent drainage from the wound resulted in re-admission to the hospital.

Patient no. 8 underwent mitral valve replacement and coronary artery bypass grafting procedures. His postoperative course was complicated by respiratory distress and atrial fibrillation on day three. On day eight, drainage from the sternotomy wound was noted as was a very unstable sternum.

Patient no. 9 underwent coronary artery bypass grafting which was followed five days later by respiratory distress and chest wall instability. There was an associated fever to $101^{o}$ F.

Patient no. 10 underwent coronary artery bypass grafting. On the eighth postoperative day, there was complete dehiscence of the sternotomy incision with a serosanguinous exudation.

## DIAGNOSIS

The diagnosis of sternotomy wound infection in each case was very obvious with serous or purulent drainage and/or sternal instability or frank dehiscence. Chest radiographs in these patients revealed mediastinal widening, mediastinal air-fluid levels and/or pleural effusions.

## PATHOLOGY

Patient no. 1 was returned to the operating room for reclosure of his dehisced sternal wound. The sternal edges were somewhat necrotic and a considerable amount of fibrinous exudate was noted in the mediastinum.

Patient no. 2 demonstrated an anterior mediastinum filled with necrotic tissue and bone. A seropurulent effusion around the heart was also found.

Free pus was found in the anterior mediastinum when patient no. 3 was returned to surgery. This was also the case in patient no. 4.

Gross pathologic finding in patient no. 5 consisted only of a massive right sided pleural effusion.

In patient no. 6 mediastinal structures were found adherent to the posterior wall of the sternum. The mediastinal spaces were without evidence of infection, but there was a great deal of fibrinous material and evidence of healing.

In patient no. 7 gross pus was observed behind the divided sternum.

There was no gross evidence of infection in the mediastinum in patient no. 8.

Patient no. 9 had a moderate amount of mediastinal fluid and blood clots.

In patient no. 10, minimal pus was noted in the mediastinum.

## BACTERIOLOGY

All but two patients had multiple organisms isolated from culture of mediastinal fluids. Staphylococcus epidermitis was the only organism isolated from patient no. 3. Culture studies in patient no. 6 were all negative, despite fever to $101^{o}$ F, leukocytosis with a shift to the left and a serous mediastinal effusion

The bacteriologic data of all patients are summarized in the following table.

| ORGANISM | NUMBER OF PATIENTS |
|---|---|
| Staphylococcus epidermitis | 8 |
| E. Coli | 4 |
| Staphylococcus aureus | 4 |
| Enterococcus species | 4 |
| Enterobacter species | 4 |
| Citrobacter species | 3 |
| Candida albicans | 2 |
| Klebsiella species | 2 |
| Streptococcus bovis | 2 |
| Acinetobacter anitratus | 2 |
| Pseudomonas aeruginosa | 1 |
| Fusobacteria | 1 |
| Proteus mirabilis | 1 |
| Streptococcus viridans | 2 |
| Lactobacillus | 1 |

TREATMENT

Treatment of patients in this series was in many respects the same in each. However, there is one important difference; about one-half of patients received postoperative irrigations of the mediastinum with an antimicrobial agent; the other half received no such irrigations. The specifics of treatment in each patient follow.

Patient no. 1 underwent copious lavaging and excision of necrotic tissues of the mediastinum. An irrigation catheter was inserted into the anterior mediastinum and was brought out through a stab incision in the skin. Two sump tubes were place inferior to the irrigation catheter; one tube was placed in the anterior mediastinum and the other was placed in the pericardium. Both were externalized via stab incisions in the skin. Postoperatively, the mediastinum was continuously irrigated with two grams of Keflin and 500 mg. of Kanamycin per liter of 0.9 mEq saline. The irrigation rate was 50 cc per hour. In order to avoid Kanamycin toxicity, the irrigant was changed to a 5% solution of Betadine in normal saline on the third postoperative day. Parenteral broad spectrum antibiotics were administered simultaneously.

Patient no. 2 underwent debridement of necrotic sternal and mediastinal tissues, followed by drainage and irrigation of the mediastinum with a Kanamycin solution and then Betadine. Two irrigation tubes were inserted into the superior end of the sternotomy wound in unspecified locations within the mediastinum. Two drainage tubes were placed in the inferior end of the wound; one was placed in the anterior mediastinum and one beneath the heart. Each irrigation cather was infused with a 5% solution of Betadine in normal saline at a rate of 50 cc per hour. Broad spectrum antibiotics were also administered parenterally.

Patient no. 3, likewise, underwent debridement of the sternotomy wound and placement of irrigation and drainage tubes in the mediastinum. She then received continuous mediastinal irrigation with a 0.5% solution of Betadine in normal saline at a rate of 50 cc per hour. Parenteral broad spectrum antibiotics were also given.

Patient no. 4, in contrast, underwent debridement and reclosure of the dehisced wound as did the foregoing patient, but no catheters were placed for postoperative irrigation. Betadine dressings were applied topically to the closed wound and broad spectrum antibiotics were administered.

Patient no. 5 received the same therapy as did patient no. 4.

Patient no. 6 underwent debridement of the mediastinum followed by irrigation with kanamycin solution and then a Betadine solution. Irrigation and drainage tubes were inserted superiorly and inferiorly in the mediastinum, respectively. The wound was reclosed and continuous irrigation with a 0.5% solution of Betadine in Ringer's lactate at 50 cc per hou.

Patient no. 7 underwent debridement of the sternal edges and the mediastinum, followed by irrigation of the mediastinum with a kanamycin solution and then Betadine. The wound was reclosed and packed with Betadine gauge. A hemovac catheter was inserted into the mediastinum via the infraclavicular area. Parenteral keflin was administered postoperatively.

In patient no. 8, there was no gross evidence of mediastinal infection. Therefore treatment was limited to irrigation of the mediastinum and reclosure of the sternal wound. Broad spectrum antibiotics were administered parenterally.

Patient no. 9 underwent debridement of the mediastinum followed by irrigation with warm saline. A mediastinal drainage tube was inserted and the wound reclosed. Parenteral cephalosporine were administered postoperatively.

Patient no. 10 experienced complete sternal dehiscence on three separate occasions, during which times he was treated with parenteral antibiotics. After the third episode closure was not attempted. Instead, wound debridement and tracheostomy were performed. Irrigation and drainage tubings were inserted into the mediastinum. The wound was left open to heal by secondary intention. The mediastinum was continuously irrigated with a 0.5% Betadine solution and broad spectrum antibiotics were administered parenterally.

MORBIDITY AND MORTALITY

Three of the 10 patients (30%) with median sternotomy wound infections died during their hospitalization.

Patient no. 1 developed Enterococcus spesis during his illness. Prior to the spesis, Enterococcus was one of several organisms isolated in culture of mediastinal fluids. A tracheostomy was performed on the sixth postoperative day to assist in the patients respiratory care. The patient recovered slowly and discharged from the hospital two months after the day of admission.

Patient no. 2 followed a course complicated by gastrointestinal bleeding and pulmonary embolism with possible pulmonary infarction. The patient followed a prolonged hospital course which ended in cardiopulmonary arrest 10 weeks after admission.

Patient no. 3 developed sustained congestive heart failure immediately postoperative. Staphylococcus aureus sepsis developed. The organisms was, again, one of several organisms cultured from mediastinal fluids. Renal failure developed which required peritoneal dialysis. The patient followed a steady downhill course to death five weeks after admission.

Patient no. 4 was dischared eight weeks after admission without complications.

Patient no. five's course was complicated by gastrointestinal bleeding on day 20 after his original surgical procedure. On the 41st postoperative day, an air-fluid level was noted in the mediastinum which gradually resolved over one month's time. The patient was discharged on the 73rd postoperative day.

Patient no. 6 had an uneventful hospital course following treatment for mediastinitis and was discharged six weeks after the day of admission.

Patient no. 7 followed a benign course to recovery from her mediastinal infection.

Patient no. 8 developed upper gastrointestinal bleeding which failed to respond to treatment and resulted in death three months after admission. Autopsy findings were remarkable for pleural peritoneal effusions, focal bronchopneumonia, severe tracheobronchitis and severe generalized atherosclerosis.

Patient no. 9 followed a benign course to recovery and was discharged three weeks after admission.

Patient no. 10 likewise followed a benign course to recovery and was discharged five weeks after admission.

## SPONTANEOUS RUPTURE OF ESOPHAGUS

Four cases of spontaneous rupture of the esophagus were reported at the Yale-New Haven Hospital between April, 1971 and June, 1977. This entity represents 20% of all cases of acute mediastinitis occurring at Yale during the specified timespan.

TABLE 1: DEMORGAPHIC DATA

| No. | Patient | SEX | Age (Yrs.) |
|---|---|---|---|
| 11 | W.O. | M | 59 |
| 12 | W.C. | M | 38 |
| 13 | R.P. | M | 60 |
| 14 | A.T. | F | 71 |

## CLINICAL SYMPTOMS

Patient no. 11 experienced severe left chest pain, supraclavicular subcutaneous emphysema, fever to $103^{o}$ F and shortness of breath as the manifestation of esophageal rupture and subsequent mediastinitis occurring after an episode of vomiting.

Patient no. 12 experienced sharp substernal chest pain, subcutaneous emphysema of the chest wall and fever to $102^{o}$ F, all of which occurred, again, after an episode of vomiting.

Patient no. 13, after several episodes of vomiting, wretching and "sudden collapse," spent five weeks in a hospital with the misdiagnosis of pneumonia. After transfer to Y.N.H.H., the only clinical sign was severe cachexia.

Patient no. 14 presented with precordial chest pain and temperature to $101^{o}$ F following five episodes of hematemesis.

DIAGNOSIS

On radiographs of the chest, three of the patients had pleural effusions and one patient had a large pleural empyema. Two of the patients had evidence of both subcutaneous and mediastinal emphysema on chest radiographs; one patient had evidence of retrocardiac air-fluid levels.

After swallowing contrast material, only three patients show evidence of extravasation of contrast outside the esophagus. The fourth patient had normal radiographic studies of the esophagus.

PATHOLOGY

In only three of the four patients were esophageal tears found at surgery. In patient no. 1, a tear in the lower left side of the esophagus was demonstrated at surgery with a large amount of cloudy fluid in the mediastinum in the region of that tear.

Patient no. 12 had a 2 cm tear in the esophagus in an unspecified position at the level of the diaphragm with copious fibrin in the mediastinum.

In patient no. 13, a left sided esophageal tear just above the diaphragm was demonstrated. Associated findings were a collapsed left lower lobe of the lung which was densely adherent to the mediastinum by fibrous tissue, minimal pus was noted in the pleural cavities.

Although patient no. 14 had demonstrated extravasation of contrast outside the esophageal lumen, at surgery, no esophageal tear was evident. The esophagus was thickened and dilated and mediastinal tissues were edematous. An acute, filmy reaction between visceral and parietal pleurae was noted. No pus was found in the mediastinum.

BACTERIOLOGY

A wide variety of organisms were isolated from the mediastinum following spontaneous rupture of the esophagus. As would be expected, each patient produced multiple organism on culture study. A summary of the bacteriologic findings is provided.

TABLE 2:

| ORGANISM(S) | NUMBER OF PATIENTS |
|---|---|
| Streptococcus viridens | 3 |
| Staphylococcus epidermitis | 2 |
| Escherichia coli | 2 |
| Serratia species | 2 |
| Klebsiella species | 1 |
| Clostridium perfringens | 1 |
| Bacteroides melaningogenicus | 1 |
| Enterococcus | 1 |
| Streptococeus bovis | 1 |
| Lactobacillus | 1 |
| Peptostreptococcus | 1 |
| Peptococcus | 1 |
| Anerobic diptheroids | 1 |
| Aerobic diptheroids | 1 |
| Pseudomonas | 1 |
| Proteus | 1 |

## TREATMENT

Treatment was essentially the same in each patient. The esophagus was approached via a left thoracotomy incision in each followed by drainage and irrigation of the mediastinum. The esophageal tears were sutured closed in patients no. 11-13. Nothing was done to the esophagus in patient no. 14 since no tear was demonstrated. In each case, chest tubes were inserted for post-surgical mediastinal drainage. A feeding gastrostomy tube was inserted in patient no. 13 at the original surgery to "rest" the esophagus until it healed. Each patient was started on broad spectrum parenteral antibiotics post-surgically.

## MORBIDITY AND MORTALITY

Patient no. 11 two days postoperatively developed a leak at the site of the esophageal suture line and fistula formation. He was re-operated upon and a feeding jejunostomy was inserted. Although his clinical condition was stabilized, he experienced an unexpected cardiac arrest and died two days after the second operation.

Patient no. 12 developed a febrile course several days postoperatively and pleural effusions and mediastinal air-fluid levels on chest radiographs. About one month after the original surgery, the patient returned to the operating room where a posterior mediastinal abscess was incised and drained. Chest tubes were inserted and irrigated daily with a Betadine solution. He then followed a course to eventual complete recovery.

Patient nos. 13 and 14 had slow but uncomplicated recoveries and were discharged three months and two months after admission, respectively.

## RUPTURE OF PYRIFORM SINUS

Two cases of mediastinitis following instrument rupture of the lateral pharyngeal spaces (pyriform sinuses) were obtained from the period April, 1971 to June, 1977. This etiology accounts for 10% of the cases of mediastinitis reported in this series. A third case from the year 1967 will also be reported on.*

TABLE 3: DEMOGRAPHIC DATA

| No. | Patient | Sex | Age (Yrs.) |
|---|---|---|---|
| 15 | G.A. | F | 69 |
| 16 | P.B. | F | 68 |
| 17 | L.S.* | F | 56 |

## CLINICAL SYMPTOMS

Patient no. 15 developed respiratory distress, foul smelling sputum and fever to $105^{o}$ F on postoperative day one for an elective cholecystectomy during which there was much difficulty in intubating the patient.

Patient no. 16 developed shortness of breath, massive subcutaneous emphysema and fever to $102^{o}$ F following intubation for radium implantation for uterine cancer.

Patient no. 17 developed marked subcutaneous emphysema extending from the face to the hips, bradycardia, hypotension and fever to $101^{o}$ F following intubation for resection of an adenocarcinoma of the sigmoid colon.

## DIAGNOSIS

Signs of perforation and mediastinitis in patient no. 15 included superior mediastinal widening and pleural effusions on plain chest radiographs. After a barium swallow, contrast was observed in the esophagus, trachea and mediastinum.

Patient no. 16 had evidence of pneumomediastinum and bilateral pleural effusions on plain chest radiographs. After a barium swallow, a tear involving the right esophagopharyngeal area at the level of the first and second thoracic vertebrae with escape of contrast into the surrounding spaces.

On plain chest radiograph, patient no. 17 showed a right sided pneumothorax, pneumomediastinum and subcutaneous emphysema. A barium swallow study demonstrated a right paraesophageal and right paratracheal collection of contrast contained in the region of the cervical soft tissues and superior mediastinum, apparently arising from a defect of the right lateral border of the pharynx.

## PATHOLOGY

Pathologic findings at surgery were generally uniform between the three patients.

Patient no. 15 was found to have a superior mediastinal abscess and a right-sided serous pleural effusion. Perforation of the pyriform sinus was not demonstrated at surgery.

Patient no. 16 was found to have a barium containing fluid in the right pretracheal space in the neck. The mediastinal compartment was not entered.

Patient no. 17 demonstrated a barium containing compartment in the superior mediastinum. No attempt to identify and repair the defect was made.

## BACTERIOLOGY

A wide variety of bacteria were isolated from the three patients reported. Both patient nos 15 and 16 had multiple organisms isolated from culture. Remarkably, patient no. 17 produced only one species of bacteria, specifically - Enterococcus.

The mediastinal culture data are summarized in the following table.

TABLE 4:

| ORGANISMS | NUMBER OF PATIENTS |
|---|---|
| Enterococcus | 2 |
| Candida albicans | 2 |
| Staphylococcus epidermitis | 1 |
| Escherichia coli | 1 |
| Klebsiella species | 1 |
| Streptococcus viridans | 1 |
| Neisseria species | 1 |
| Aerobic diptheroids | 1 |

## TREATMENT

Patient no. 15 underwent incision and drainage of the mediastinum via the neck. A right thoracotomy with placement of a chest tube in the right pleural cavity were also performed. Postoperatively, broad spectrum antibiotics were administered parenterally.

Patient no. 16 underwent incision and drainage of the mediastinum through the right side of the neck. The mediastinum was irrigated with a kanamycin solution and large drains were left in place. Postoperatively, broad spectrum antibiotics were administered parenterally.

Patient no. 17 underwent incision of the anterior aspect of the sternomastoid muscle. The dissection was carried down to the retrovisceral space. Several large drains were left in place and brought out through the skin. Large pressure dressings were applied externally. Broad spectrum antibiotics were administered parenterally postoperatively.

## MORBIDITY AND MORTALITY

Postoperatively, patient no. 15 followed a progressive downhill course with continued respiratory distress and fever. She experienced a cardio-pulmonary arrest and died one month after admission. At autopsy, several non communicating locules of a purulent exudate were found in the lower left anterior mediastinum. Contusions of the right pyriform sinus were present, but there was no evidence of perforation. There was also acute and healing bilateral pneumonia.

Patient no. 16 had a rather benign postoperative course after drainage of her mediastinum. She did develop a right pleural effusion and hemothorax. Dhe was discharged three weeks after the day or original admission.

Patient no. 17 too, had a benign postoperative course and was discharged three weeks after the day of admission.

## INSTRUMENT PERFORATION OF THE ESOPHAGUS

Only one case of perforation of the esophagus during instrumentation could be found at the Yale-New Haven Hospital between April, 1971 and June, 1977. Additionally, two known cases of mediastinitis of this same etiology which occurred several years prior to the dates encompassed by this study are included for comparative purposes.* The clinical courses of these three patients are summarized below.

DEMOGRAPHIC DATA

| No. | Patient | Sex | Age (yrs.) |
|---|---|---|---|
| 18 | J.M. | F | 73 |
| 19 | V.B.* | F | |
| 20 | M.S.* | F | 25 |

## CLINICAL SYMPTOMS

Patient no. 18 had a history of distal obstructing carcinoma of the esophagus and was admitted for esophagoscopy. After the procedure she complained of thoracic pain, but remained afebrile until two days after the procedure when her temperature reached $103^{o}$ F.

Patient no. 19 was admitted in 1952 to undergo esophagoscopy for benign stricture of the lower esophagus. After the procedure, the patient experienced increasingly severe anterior chest pain.

Patient no. 20 was admitted to the hospital in 1956 for esophagoscopy and bougie dilatation for benign strictures of the esophagus. After recovery from anesthesia she complained of right chest and shoulder pain.

DIAGNOSIS

In patient no. 18, contrast study of the esophagus revealed extravasation of contrast nine centimeters above the gastroesophageal junction and on the left side of the esophagus. The obstructing cancer was distal to the perforation.

Patient no. 19 demonstrated cervical subcutaneous and mediastinal emphysema on chest radiographs on the first day after the procedure.

Patient no. 20 demonstrated hydropneumothorax on plain chest radiographs and contrast study of the esophagus revealed extravasation into the right pleural cavity at the level of the middle third of the esophagus.

PATHOLOGY

When patient no. 18 was returned to surgery, purulent fluid was found in the left hemithorax. The mediastinum was entered but findings were not reported.

Patient no. 19 died prior to treatment due to cardiopulmonary arrest on day two day esophagoscopy. At autopsy the pleural spaces contained black, foul fluid and pleural membranes were markedly hyperemic. The posterior mediastinum was blackened autolyzed and friable. Retrospectively, the patient's esophagoscopy procedure was made more difficult due to an underlying tortuous spinal column.

In patient no. 20 the pleural surfaces were found covered with thick mucous material. The mediastinum contained about 100 cc of bloody chyle streaked fluid.

BACTERIOLOGY

Cultures of pleural fluid from patient no. 18 grew Klebsiella, Candida and Acinetobacter anitratus.

There were no bacteriologic data available for patient no. 19.

Patient no. 22 grew Proteus and para-colon bacillus from pleural fluid. No information was available on cultures of mediastinal fluid.

TREATMENT

Patient no. 18 underwent emergency esophagogastrectomy and irrigation of the mediastinum with a Kanamycin solution. Broad spectrum antibiotics were administered postoperatively.

Patient no. 20 underwent repair of her esophagus and the thoracic duct was tied off above the diaphragm because of the possibility of damage to it. The mediastinum was drained into the right pleural space via a posterior thoracotomy. The mediastinum was irrigated with a saline solution and chest tubes were inserted for drainage. No mention of postoperative antibiotics was made.

MORBIDITY AND MORTALITY

On day seven after her first operation, patient no. 18 developed signs of an anastomotic leak which was confirmed by esophageal swallow of barium. A right-sided empyema developed which required closed thoracostomy and later open drainage of the upper right chest. The patient developed bilateral pulmonary infiltrates and respiratory distress which required endotracheal intubation. She died suddenly on day 20 after admission.

Patient no. 19, to reinterate, died of cardiopulmonary arrest prior to any treatment.

Patient no. 20 required a feeding gastrostomy to supplement her therapy, but her subsequent clinical course was benign and she was discharged one month after admission.

POSTOPERATIVE LEAKS FROM ESOPHAGEAL SUTURE LINES

Only one cases of mediastinitis fitting this category was found during the period April, 1971 through June, 1977. A second case* which occurred prior to 1971 is also included here for comparative pruposes.

No. 21: E.P., a 65-year-old female was admitted to the hospital in mid 1977 for total gastrectomy for Zollinger-Ellison syndrome and gastric ulcerations. Following her transthoracic gastrectomy, she developed chest pain, shortness of breath and daily fever with peaks to 101.4$^{o}$ F. An echocardiogram revealed a pericardial effusion and loculated fluid lateral to the cardiac silhouette on the left which were thought to represent a large collection of mediastinal fluid. A barium swallow revealed a small area of extravasation at the distal end of the esophagus. On chest radiograph an air-fluid level in the mediastinum at the level of the aortic arch and air along the left heart border were noted. These findings were consistent with an esophago-jejunal anastomotic leak. The patient was returned to surgery for open drainage of pericardial fluid and mediastinal and pleural empyemas. About 200 cc of pus were drained from multiple loculated areas within the thoracic cavity spaces. followed by copious irrigation with a Bacitracin solution and placement of chest tubes connected to pleurovac suction. A peurose drain was left in the retrocardiac space. Culture of the pericardial fluid was sterile. Culture of abscesses in the chest cavities yielded the following organisms: Streptococcus viridans, Neisseria species and Candida albicans. Broad spectrum antibiotics were ad-

ministered postoperatively. At present, the patient is suffering from staphylococeus epidermitis and straptococcus bovis septicemias and is in serious condition in surgical intensive care.

No. 22: E.G.* was admitted to hospital late in 1968 for excisional repair of a Zenker's diverticulum. On postoperative day one, massive subcutaneous emphysema at the base of the neck and a barium swallow revealed lateral extravasation of contrast in the area of the esophageal surgery. The patient returned to surgery to repair the leak but none was found although purulent material and barium were found in the area of the repaired diverticulum. Ten days later, dysphagia nd fevers to $102^{o}$ F developed along with mediastinal widening on chest radiograph. The abscess was incised and redrained through the cervical route and broad spectrum antibiotics were given postoperatively. Cultures of the abscess material yielded the following organisms: Enterobacter, Streptococcus viridans, Klebsiella, Proteus mirabilis, Enterococeus, Candida albicans and coagulase negative staphylococeus. The patient recovered and was discharged seven weeks after admission.

MISCELLANEOUS

Following are two unusual, but nevertheless important cases of acute mediastinitis.

No. 23: G.A., a 45-year-Old male, had a three year history of chronic productive coughing, cramping mid-sternal chest pain after meals, hemoptysis, recurrent submandibular, cervical and auricular swellings, solid food dysphagia and increasing hoarseness. Although not recurrent, the patient did experience fevers on several occasions as high as $102^{0}$ F. Radiographic studies revealed a retropharyngeal mass and mediastinal widening which extended down to the seventh cervical vertebra and the tracheal bifurcation, repectively. A barium swallow revealed shifting of the esophagus to the right side of the mid-line. At surgery, the neck mass was incised and drained off 75 cc of coffee-colored material. A biopsy of the posterior pharyngeal wall and necrotic debris which, on histologic study, showed extensive tissue necrosis and acute inflammation. There was no evidence of tumor. The aspirate of the pharyngeal mass grew out the following on culture: E. coli, Staphylococcus epidermitis, β-streptococcus, ∝ streptococcus and enterococci. Acid-fast studies were negative. The patients symptoms subsided after drainage of the mass and he was discharged three weeks after admission. The cause of his mass was never determined.

No. 24: B.G., a 65-year-old female, developed infection at the site of an epicardial pacemaker, nine months after its implantation. Chest radiographs revealed spontaneous gas formation along the sternum which was consistent with sternal abscess. At surgery, a pocket of pus was found behind the sternum with lateral extension towards the left. The wound was irrigated with saline and packed with betadine sponges. Broad spectrum antibiotics were administered postoperatively. Culture studies of the anterior mediastinal abscess grew out Proteus mirabilis and staphylococcus epidermitis. She was discharged six weeks after admission.

## SUMMARY

A total of 24 cases of acute mediastinitis are reported. However, three of these predate the period April, 1971 to June, 1977 and so are not included in the following summary and discussion.

Table 1 summarizes the etiologies, incidence and mortality data and Table 2 summarizes the bacteriologic data.

## ETIOLOGY AND INCIDENCE

Median sternotomy wound infections were by far the most common causes of acute mediastinitis in this series (48%). This is in contrast to Neuhof's 1936 series[41] of 66 cases in which he reported esophageal perforation by instrumentation to be the most common etiology of mediastinitis (50%). In a more modern report, Enquist et al.[9] likewise reported instrument perforation of the esophagus as the most common etiology of mediastinitis, followed in frequency by median sternotomy wound infections. The differences between this present series and those referred to in the literature probably represent a combination of the following factors: (1) the advent of fiberoptic esophagoscopes; (2) the increasing numbers of open heart surgeries.

## BACTERIOLOGY

Various combinations of 25 different organisms were isolated from the 21 patients in this study. Staphylococcus epidermitis was the most common isolate occurring in two-thirds of the cases. The predominance of this non-invasive inhabitant of the skin is not at variance with the findings of Korblum and Osmond[29] who reported in 1934 that the streptococci and the staphylococci are the most common organisms isolated from patients with acute mediastinitis.

TABLE 1:

ETIOLOGY/INCIDENCE/MORTALITY

| ETIOLOGY | # OF CASES/% OF ALL CASES | #OF DEATHS/% OF ALL CASES |
|---|---|---|
| Median Sternotomy Wound Infections | 10/(48%) | 3/(21%) |
| Spontaneous rupture of esophagus | 4/(19%) | 1/(5%) |
| Instrument rupture of pyriform sinus | 3/(14%) | 1/(5%) |
| Instrument perforation of esophagus | 1/(5%) | 1/(5%) |
| Miscellaneous | 2/(10%) | 0 |
| Post-esophageal surgery anastomotic leak | 1/(5%) | 0 |
| TOTALS | 21/(100%) | 6/(29%) |

TABLE 2:

BACTERIOLOGY

| ORGANISMS | NUMBER OF PATIENTS |
|---|---|
| Staphylococcus epidermitis | 14 |
| E. coli | 8 |
| Enterococcus | 7 |
| Streptococcus viridans | 7 |
| Candida albicans | 6 |
| Klebsiella | 5 |
| Enterobacter | 4 |
| Staphylococcus aureus | 4 |
| Proteus | 3 |
| Citrobacter | 3 |
| Acinetobacter anitratus | 3 |
| Streptococcus bovis | 3 |
| Neisseria | 3 |
| β-Streptococcus | 2 |
| Pseudomonas aeuriginosa | 2 |
| Bacteroides melaningogenicus | 2 |
| Lactobacillus | 2 |
| Aerobic diptheroids | 2 |
| Serratia | 2 |
| Fusobacterium | 1 |
| clostridium perfringens | 1 |
| Peptostreptococcus | 1 |
| Peptococcus | 1 |
| Aerobic diptheroids | 1 |
| ∝ - streptococeus | 1 |

## SYMPTOMS, SIGNS AND DIAGNOSIS

Though symptoms and signs of acute mediastinitis varied among patients in this series, there are no changes to be reported in comparison to the world's literature. Neither has the uses of the chest radiograph been surplanted as highly efficacious diagnostic aides.

## TREATMENT

The cornerstone of acute mediastinitis of all etiologies continues to be some form of surgical drainage and irrigation of infected foci and postoperative antibiotics. A number of variations on this basic therapy is noted in this series, each of which proved effective in the outcome. Unfortunately, there are no controlled studies in the literature which indicate that any one method is more efficacious than another.

## MORBIDITY AND MORTALITY

Nearly 50% of the 21 cases reported here were associated with some degree of morbidity occurring after treatment of the mediastinitis. The mortality in this series was 29% (6 cases).

## CONCLUSIONS

Acute mediastinitis is a relatively rare disease which is most commonly caused by postoperative median sternotomy wound infections. Signs and symptoms varying according to etiology. The chest radiograph is often the most useful diagnostic aide. A wide variety of infecting organisms are found, but the staphylococcus organism is most common, irrespective of etiology. Treatment, which consists of surgical drainage and debridement of infected foci, is effective but morbidity and mortality of this disease is relatively high. A goal for the future, then, should be to lower the morbidity and mortality of this disease with improvement of existing therapies.

# V. CHRONIC MEDIASTINITIS

## MATERIALS AND METHODS

Retrospectively, all cases of chronic mediastinitis were sought out via an intensive medical records search of the medical records library at the Yale-New Haven Hospital over the period April, 1971 to June, 1977. The following classifications were relied upon to identify cases: (1) Mediastinitis (519.9); (2) Histoplasmosis (115.0, 115.1, 115.2, 115.9); (3) Syphyllis (095); (4) Tuberculosis (012.9) and (5) Actinomycosis (113).

Additionally, members of the following departments were interviewed to determine if additional cases of chronic fibrous mediastinitis could be found: (1) Cardiothoracic Surgery, (2) Infectious Diseases and (3) Radiation Therapy. All records received were reviewed for diagnosis of mediastinal fibrosis and its associated signs and symptoms, radiographic findings, bacteriology, pathology, treatment and morbidity and mortality.

RESULTS

Only one case of chronic, fibrosing mediastinitis was found between the period April, 1971 and June, 1977. A second case* was found which predates the period under study and is included for comparative purposes.

No. 1: V.A. was a 48-year-old female who was admitted to the hospital for severe and increasing dyspnea on exertion. She had a five year history of right chest pain and hemoptysis in association with a right pleural effusion and a right upper lobe cavitary lesion found at surgery to be nonspecific fibrosis. Symptoms of congestive failure and dyspnea on exertion began one year prior to admission. There was also a one year history of a cardiac murmur.

An echocardiogram revealed right ventricular enlargement. Cardiac catheterization revealed an absent right pulmonary artery segment and high-grade branch stenosis of the left pulmonary artery. A perfusion lung scan revealed no perfusion of the right lung.

At surgery, dense adhesions were encountered around the take-off of the left and right pulmonary arteries. The superior vena cava was enlarged and surrounded by dense fibrous tissue in the mediastinum. Dense fibrous tissue completely surrounded the right pulmonary artery. Numerous large succulent mediastinal lymph nodes were encountered and removed. Some of the mass was resected, but the fibrosis was so extensive and infiltrative that local anatomy was obliterated beyond recognition.

The lymph nodes removed at surgery were remarkable for chronic lymphadenitis. Fibrotic material were sterile.

Cryptococcus titres and sputum studies for acid-fast bacteria were both negative.

Complete blood count was normal.

The patient was discharged with a guarded prognosis.

No. 2:* B.F. was a 57-year-old female admitted for diabetic ketoacidosis. After admission she was found to have a left lower lobe pneumonia, hyper-thyroidism and a probable small bowel infarction. There were associated fevers

to $105^{o}$ F, leukocytosis, tachycardia and insulin resistance.

Additionally, there was marked jugular venous distention with a central venous pressure of 210 mm of water. Chest radiograph showed marked calcification of the pericardium and left coronary artery.

The patient died suddenly of a cardiac arrest. Autopsy findings included sclerosing mediastinitis involving the left mediastinum and hilum of the left lung with complete obstruction of the left main pulmonary artery, marked left pleural fibrous adhesions and fibrinous pericarditis. There were no reports on cultures of fibrous tissues.

## SUMMARY AND CONCLUSIONS

Discussed are two cases of chronic, fibrosing mediastinitis, only one of which occurred within the last six years. Both cases fit into Yacoub's and Thompson's[59] classification of type I pulmonary hilar fibrosis.

The realm of fibrosing disease of the mediastinum remains an enigma in terms of the lack of progress which has been made over the years in understanding the pathogenesis of this disease. Fortunately, while we await a breakthrough in the latter, fibrosing mediastinitis remain a rarely encountered medical curosity.

FIGURES

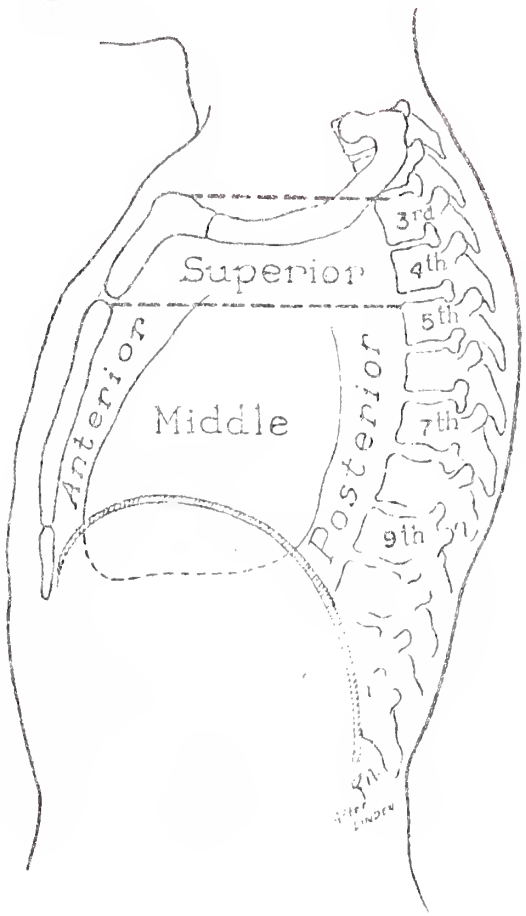


Figure 1: The mediastinum on left lateral projection. [31]

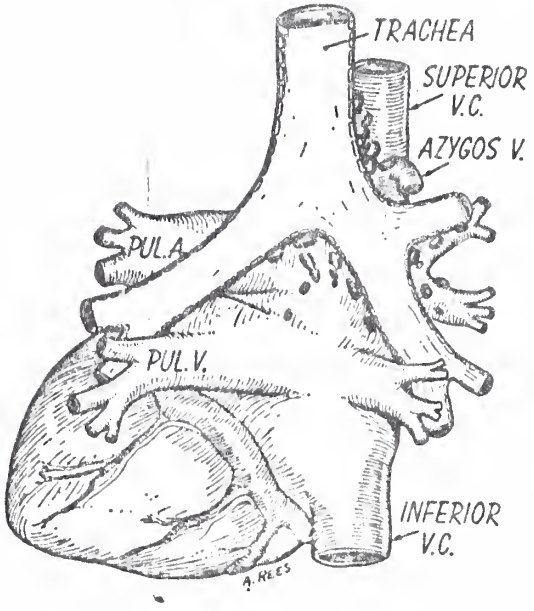


Figure 2: Posterior view of mediastinum showing anatomical relationship of mediastinal lymph nodes to various mediastinal structures.[17]

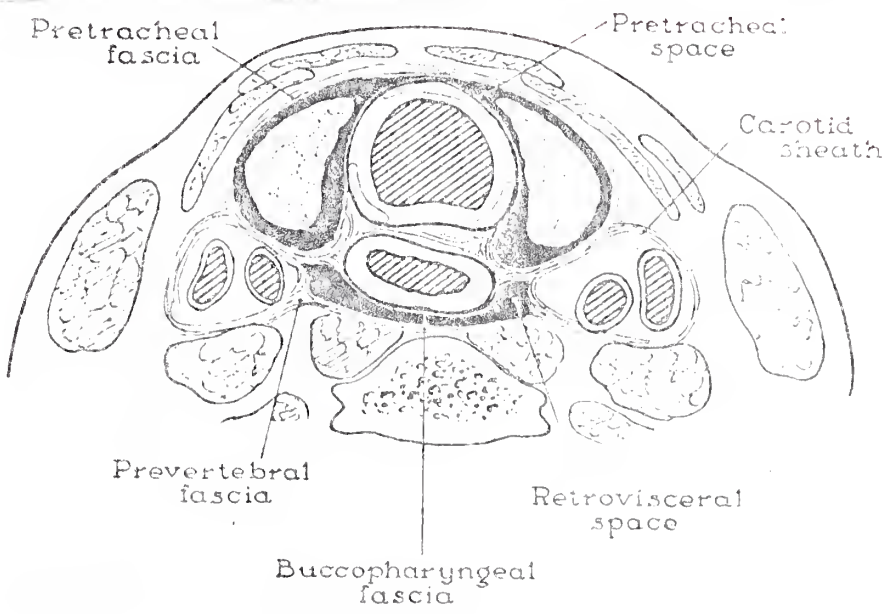


Figure 3: Cross-section of neck indicating major fascial planes and spaces.[44]

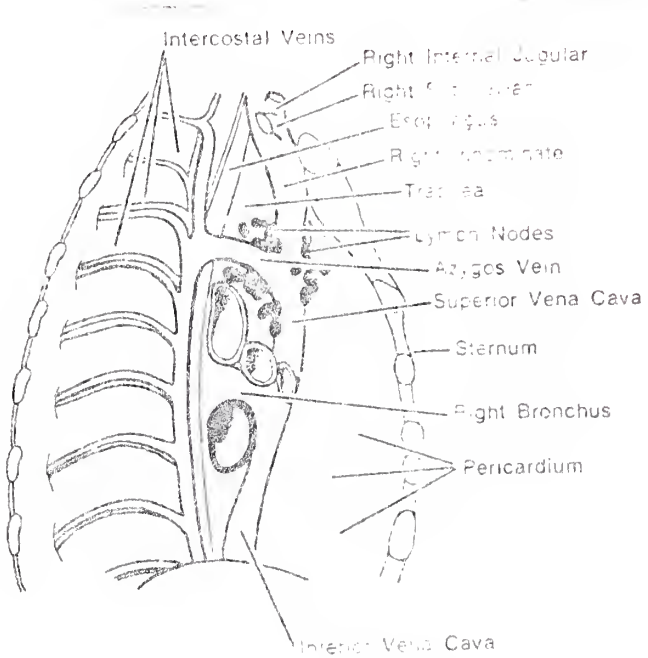


Figure 4: Schematic representation of right sagittal section of the thorax showing encasement of the superior vena cava by lymph nodes.

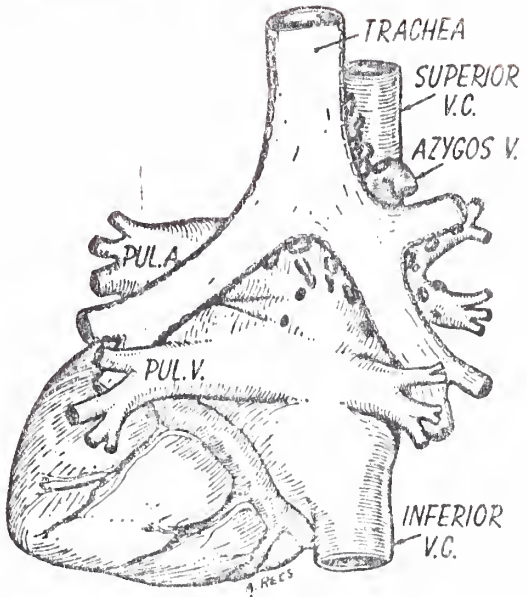


Figure 5: Posterior view of mediastinum showing anatomical relationship of mediastinal lymph nodes to various mediastinal structures.

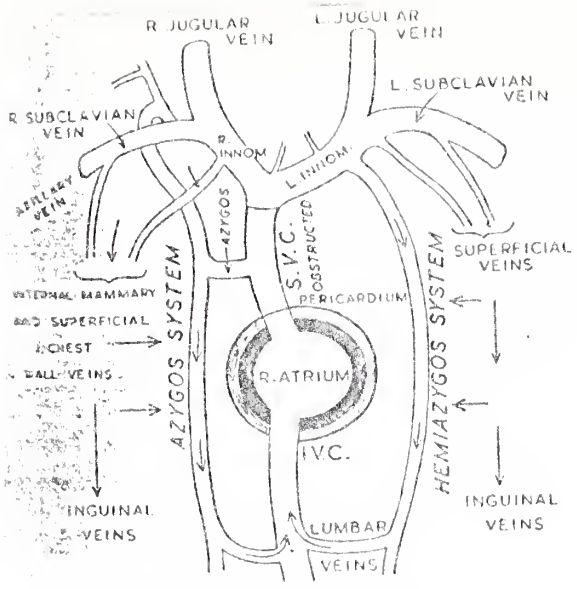


Figure 6: Obstruction of the superior vena cava and the azygous venous system.[2]

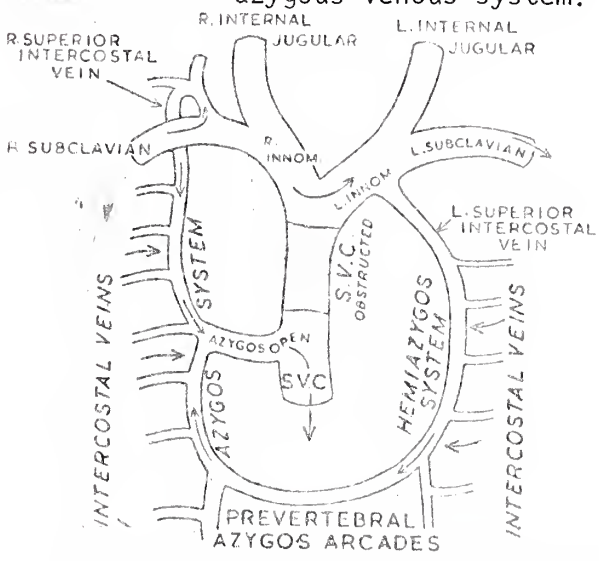


Figure 7: Obstruction of the superior vena cava.[2]

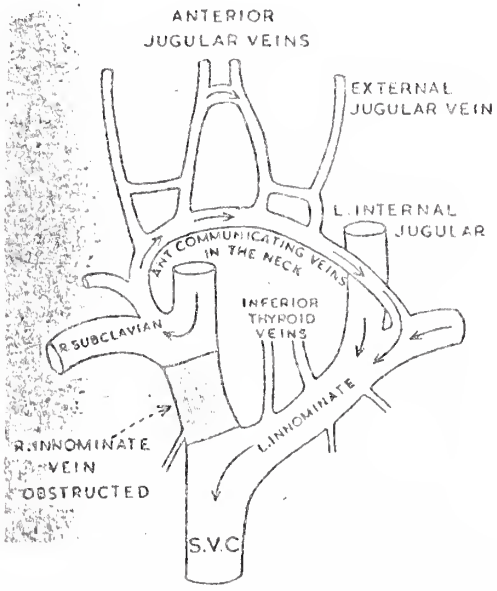


Figure 8: Obstruction of the right innominate vein.[2]

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